CASE 5

OF

HEMIPLEGIC EPILEPSY

FOLLOWING

FRACTURE OF THE SKULL.

BY

CHARLES B. BALL, M.D., F.R.C.S.I.,

SURGEON TO SIR PATRICK DUN'S HOSPITAL,

ETC., ETC.

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For the notes of this case of hemiplegic epilepsy following fracture of the skull, I am indebted to my dresser, Mr. W. Kiddle :—

Case.—M.G., aged thirty-six, a well-built, powerful man, who had been a policeman, but was dismissed the force for intemperance, and was now by occupation a car-driver, was admitted into Sir P. Dun's Hospital on Friday, April 27, 1883. I had seen him that morning at his lodging, where I elicited the following statement of his case :—On the previous Monday (23rd inst.) he was driving a car at Donnybrook, when the horse stumbled and he fell off on his head. He was stunned for a few minutes, but then got up and drove his fare back to Jury's Hotel, where, after imbibing some of the national restorative, he said he felt quite well. On Tuesday, Wednesday, and Thursday, he was about at his work as usual, but on Friday morning he was found insensible in his bed by some neighbours who went to see why he had not come to his work. He lived in a room by himself. Shortly after he was found it was stated he had a fit.

Upon admission his head was shaved, and during the process he had a convulsion which affected the left side of the face and arm only. A detailed examination of his head showed an abrasion of the skin close to the left parietal eminence. This did not even expose the aponeurosis of the occipito-frontalis muscle, and no irregularity of surface of the bone could be felt in the immediate neighbourhood. The only other condition to be detected on the outside of the skull, which could in any way be considered abnormal, was an irregularity and ridge in the neighbourhood of the lambdoidal suture on the left side, but over this there was not the slightest superficial sign of injury. The left arm and leg were decidedly weaker than the right, although he was able to move them to some extent. There was slight convergent strabismus. He was in a condition in which it was impossible to get any information on subjective

phenomena. When roused he would answer questions much as a drunken man, in language not of the choicest. During the first twenty-four hours he was in hospital he had four epileptiform convulsions affecting the left side of the face and the left arm. Early on the morning of Sunday, 29th, the fits became very much more frequent, and when I visited the hospital about 10 30, a.m., I saw several of the attacks, and they invariably commenced in the same way. The eyes first wandered off to the right side and became fixed for a few seconds, then nystagmus, with turning of the eyes to the left side, and convulsions of the facial muscles came on, and about the same time the upper extremity became affected. The spasms did not appear in the lower extremities for several seconds after they were well established in the arm and face. The average duration of each fit was about three-quarters of a minute, the longest noted being $1\frac{1}{4}$ minutes. There was no period of coma after the convulsions. He would talk as rationally immediately after the fit as he did before, and sometimes told us he had just been for a drive. There was complete paralysis of the left upper and lower extremities, with partial facial palsy on the same side. Reflex action was well marked. When his left hand was lifted and placed in his right he said that it belonged to the man in the next bed. During the 30th and 1st the convulsions were very frequent, occurring every few minutes, but during the 2nd they decreased very much in frequency, and he died comatose on the morning of May 3.

The question of operative interference was frequently discussed during his illness, but we determined that no operation was justifiable, as all the indications of brain lesion were on the side opposite the superficial injury, and the result of the *post mortem*, as will be shortly detailed, showed how futile any attempt at trephining would have been. Treatment was mainly directed to trying to moderate the force and frequency of the convulsions by large doses of chloral and bromide of potassium.

At the *post mortem* I had the advantage of the help of my colleague, Dr. T. E. Little. On raising the scalp we found a sub-aponeurotic extravasation of blood over the whole surface of the cranium. Of anything this was rather less marked under the scalp wound than at other parts. Corresponding with the ridge felt during life was a fissure which traversed the left lambdoidal suture, and round this the extravasation was sub-pericranial. On raising the calvaria we found blood effused between the bone and dura mater over a considerable extent, which was distributed evenly on both sides of the middle line, and extending over the upper portions of the occipital and the inferior posterior portions of the parietal regions. There was a large collection of blood in the cavity of the arachnoid on the right side covering the whole of the right cerebral hemisphere, and upon removal of the brain we found three distinct areas of brain lesion on the right side—viz., the inferior

frontal convolution, the temporal convolutions, more particularly the middle, and the inferior occipital convolution. In these situations the cortical substance was much lacerated and soaked with extravasated blood, and the pia mater and visceral arachnoid were torn through. In addition to these injuries there were several small hæmorrhages underneath the visceral arachnoid on the same side. There was no lesion to be found on the left side or in the interior of the brain. Returning now to the skull and tracing the fracture, we found three lines radiating from a point about two inches to the right of the middle line in the lambdoidal suture, and from this focus one line passed round by the suture to the left side and then up into the parietal bone, but not as far as the eminence; a second passed through the right parietal bone to the groove for the middle meningeal artery, but its termination was lost in the saw cut for removal of the brain; a third and shorter one passed down the occipital bone in the posterior fossa of the skull. The external aspect of the fracture gaped more than the internal, and at one point the outer table was slightly splintered.

This case presents for consideration many points of pathological and clinical interest; and first we may consider the mechanical conditions under which the injuries were produced, and in doing so we must bear in mind the following three signs of injury—viz., 1. External evidence of injury applied to the *left* parietal eminence. 2. Extensive lacerations of the cortical gray matter of the under-surface of the *right* cerebrum, the largest area of disintegration being diametrically opposite the left parietal eminence. 3. A fracture, the focus of which was at an angle of 90° with a line joining the point of external injury and the area of greatest brain lesion.

That the brain should be lacerated by *contre-coup* in injuries of the head is a fact that has been very frequently observed—indeed, according to Bergmann, it is the rule to find in cases where a blow is disseminated over a large area of the head, as when a person falls on hard ground, that the greatest brain lesion is observed at a point opposite the part struck, and it is only in those cases where the blow is limited in area that injury of the brain corresponds with the part struck. As the vast majority of injuries are inflicted on the upper half of the cranium, so we find the under-surface most frequently lacerated. This fact was observed by Brodie, who attempted to explain it by the harder, more resisting, and uneven surface of the base of the cranium. M. Duret attempts to account for these injuries by waves which pass in every direction from the point struck, and which, meeting at the opposite side of the head, cause the disruption of the brain.

This theory is open to some objections. In the first place, the space occupied by the cerebro-spinal fluid is too small and too obstructed by the points at which the convolutions touch the arachnoid for any very powerful wave to pass; and, even if we granted that such waves could pass freely, it could not account for the fact that a *greater* amount of injury is inflicted at the opposite side than at the side struck. M. Gama made a number of experiments with glass globes filled with gelatine, and he found, when the vessel was struck a severe and diffused blow, the gelatinous mass separated from the glass to a greater extent at the opposite side, and this fact, although he does not appear to have followed it up, gives, I think, the key to the explanation of injury of the brain by *contre-coup*. When a person falls heavily on the top of the head the brain is compressed towards the part struck, and separates itself from the base of the skull—a vacuum is therefore left. The internal vascular pressure in the brain is consequently unopposed, and at the same time is increased by the pressure to which the mass of the brain is subjected, and as a result the smaller vessels give way, breaking down the soft gray matter and tearing through the arachnoid, exactly in the same way that we see ecchymosis forming under a cupping glass. A point which was observed in the case above related favours this theory—namely, that the disruption of the gray matter extended down into the sulci between the convolutions, which manifestly excludes any question of pressure against the bone. It is, of course, impossible to test this theory satisfactorily by experiments on the dead body, as the important element of vascular pressure is absent.

My colleague, Dr. Purser, kindly drew my attention to a paper by v. Wahl, on fracture of the base of the skull, where he explains certain fractures, in which there is breaking of the skull outwards at an angle of 90° from the part struck. When a semi-elastic sphere is compressed at the poles there is a tendency to a break outwards at the equator, and when a person falls on the head a similar compression takes place between the weight of the body, or even the weight of the head, and the part which is struck. We can easily perform a simple experiment which will more clearly explain this matter. If an orange is thrown forcibly against a white-washed wall we find that the part which strikes the wall can be identified by the white mark, and if it has been thrown with sufficient force to rupture the orange, it will be found that the centre of the rupture does not correspond with the white mark, but

is at an angle of 90° with it. The line of rupture is, however, in the direction of the white mark. Now this appears to have been exactly what happened in the above case—the point of impact was the left parietal eminence, the focus of fracture in the right lambdoidal suture, and a fissure extended in the direction of, but not as far as, the left parietal eminence. Against this theory it may be urged that the fracture was starred, but when we consider the irregularities present in the suture we can readily imagine how a line of fracture might divide into two; and the gaping of the external aspect of the fracture, together with the slight comminution of the external table, affords evidence, I think, that the fracture occurred from within outwards.

The most interesting clinical features of this case were the epileptiform convulsions, which corresponded closely with the condition so ably described by Hughlings Jackson as indicating lesion of the cortical gray matter, and now well known by the name of Jacksonian epilepsy. The three important points of this affection were very well marked:—(1) The spasm beginning in one group of muscles and gradually spreading in regular order to the other muscles of the same side—in this case the protospasm was invariably in the eyes; (2) Paralysis of the affected muscles; (3) The slight degree of insensibility.

For the production of this form of epilepsy mere destruction of the cortical substance is not sufficient; secondary changes in the neighbourhood of the injured part appear to be necessary—hence the interval which elapsed between the injury and the onset of the convulsions.

The experiments of Hitzig show that removal of a portion of the cortical surface of the cerebral hemisphere in animals was followed by epilepsy after periods varying from one day to five weeks; and for the production of these convulsions inflammation does not appear to be essential, and certainly was not present in this case. The changes necessary to repair of the injury would seem in some instances to be sufficient.